What would you tell other people?

If you start to feel like you need to lose weight and you're taking it to extremes, go and find someone to talk to. This eating disorder can and will kill you. If you have an eating disorder, seek help now, before it's too late. The longer you live with an eating disorder, the harder it is to give it up. Furthermore, I think people need to know that:

- People with eating disorders aren't just trying to get attention
- People with eating disorders use their eating disorders to cope with stress
- Getting better and becoming healthy is an everyday battle, as you know that you can relapse and have to start all over.

If you know anyone who you think has an eating disorder, try to talk to them in a way that they won't think they are being ganged up on, and talk to a person you feel like you can trust. I wish I had sought treatment early on.

Lesson of the week

Poor glycaemic control caused by insulin induced lipohypertrophy

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Good glycaemic control reduces the risk of complications of diabetes.[1] Secondary failure of oral hypoglycaemic treatment is common in patients with type 2 diabetes, and thus insulin treatment is often needed to improve glycaemic control. Indeed, in the UK prospective diabetes study 38% of patients with type 2 diabetes needed insulin treatment after 10 years.[1] The reasons for poor glycaemic control are many; and despite insulin treatment many patients with diabetes have poor glycaemic control.[2] Lipohypertrophy is characterised by a benign "tumour-like" swelling of fatty tissue secondary to subcutaneous insulin injections. We describe two cases in which poor glycaemic control was directly related to insulin induced lipohypertrophy, recognition of which led to major improvements in glycaemic control.

Case reports

Case 1

A 37 year old woman had been given a diagnosis of type 1 diabetes when she was 7 years old. She was treated with soluble insulin twice daily. She was transferred to our diabetes unit in 2000. She had experienced problems with fluctuating blood glucose concentrations, recurrent hyperglycaemia, and frequent unpredictable hypoglycaemia, despite compliance with diet and regular self monitoring of blood glucose. At her most recent annual review she was noted to have mild background retinopathy but no other microvascular or macrovascular complications of diabetes. Results of lipid, urea and electrolytes, and thyroid function tests were normal, but her glycated haemoglobin was 9.1% (normal range 3.6% to 5.1%). She was treated with Human Actrapid (Novo Nordisk) at a total daily dose of 30 units a day and Human Insulatard (Novo Nordisk) 26 units at night, both administered through a pen device. After her latest annual review she was referred to the diabetes specialist nurse for further follow up in an attempt to improve her glycaemic control.

The diabetes specialist nurse noted that she had fluctuating blood sugar concentrations, ranging from 2.0 mmol/l to 18.9 mmol/l, and unpredictable hypoglycaemic episodes occurring three or four times a week, with good awareness. The nurse reviewed her injection technique and noted significant lipohypertrophy at the sites of her abdominal injections. She was advised to avoid these sites for future injections and to reduce her insulin dose by 10%. Over the next six months her glycaemic control improved, with home tests showing blood sugar concentrations ranging from 3.4 mmol/l to 9.8 mmol/l before meals and 6.7 mmol/l to 12.3 mmol/l after meals. The frequency of occurrences of hypoglycaemia reduced to less than once a month. Her glycated haemoglobin fell to 6.8% within three months, despite the reduction in insulin dose.

Case 2

A 56 year old woman was seen in our diabetes clinic with poor control of her diabetes. She had been given a diagnosis of type 2 diabetes 10 years earlier and was changed to insulin treatment four years earlier because of secondary failure of oral hypoglycaemic treatment, when her glycated haemoglobin was 9.5%. She was treated with Human Mixtard 30 insulin (Novo Nordisk) twice daily at a total daily dose of 64 units and achieved reasonable control of her diabetes within six months of insulin treatment (glycated haemoglobin 6.4%). At her most recent annual review, however, it was noted that her glycaemic control was deteriorating—with home blood sugar concentrations ranging between 3.8 mmol/l and 24.5 mmol/l and occasional unpredictable hypoglycaemia. Her treatment was changed to Human Actrapid and Human Insulatard in a basal bolus dosing regimen at a total daily dose of 52 units a day. Despite good dietary compliance, regular self monitoring of blood sugar concentrations, and adjustment of her insulin dose she still had erratic glucose concentrations and poor control, with a haemoglobin A_{1c} of 8.9%. There was no evidence of microvascular or macrovascular complications of diabetes, and lipid, urea and electrolytes, and thyroid function tests all gave normal results.

She was seen by a diabetes specialist nurse who reviewed her injection technique and injection sites and noted that she had severe lipohypertrophy. She

Look for lipohypertrophy in all patients being treated with insulin, particularly in patients with erratic glycaemic control

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was advised to avoid injecting into these sites, to rotate injection sites, and to reduce her insulin dose by 10%. Within three months her glycated haemoglobin fell to 6.7%, her blood sugar concentrations improved to between 4.5 mmol/l and 10 mmol/l, despite her lower dosage of insulin, and she had no further episodes of hypoglycaemia.

Discussion

Diabetic lipodystrophies, particularly lipoatrophy, were common local complications of insulin treatment in patients treated with bovine or porcine insulins.[3] With the introduction of human recombinant insulins, lipoatrophy has become uncommon, but lipohypertrophy remains a major problem. It is estimated that the prevalence of clinically significant lipohypertrophy is around 20% to 30% in patients with type 1 diabetes and around 4% in patients with type 2 diabetes.[4]

Lipohypertrophy seems to be due to a cellular response of adipocytes to the local effects of injected insulin. However, susceptibility to the complication varies significantly, and hence immunological factors may be important. A recent study of lipohypertrophy in children and young adults with type 1 diabetes found that titre of insulin antibodies correlated directly with the degree of lipohypertrophy in these patients.[5] Other suggested risk factors for lipohypertrophy include frequent injection at the same site, type of insulin, number of injections a day, total daily dose of insulin, reuse of needles, and use of pen devices rather than syringes.[4]

Injection into lipohypertrophied injection sites can lead to problems with glycaemic control. Evidence indicates that insulin absorption can be significantly delayed, leading to erratic glycaemic control and unpredictable hypoglycaemia.[6] The lipohypertrophied areas can be unsightly, and the only available treatment for the condition is liposuction, although not injecting into the sites may reduce their size over time.

Diabetes UK suggests that injection sites be examined each year for evidence of lipohypertrophy as part of patients' annual diabetes review.[7] Although our patients were asked about injection sites at their annual review, the lipohypertrophy was not found on routine clinical examination. It was only when a nurse specifically looked for lipohypertrophy that it was found. Ideally, sites should be palpated rather than just visually examined. Sites are often asymmetrical, as the dominant hand tends to inject into one side preferentially. Advice to patients when they start insulin treatment to rotate sites is mandatory. Once sites of hyperlipotrophy are identified, avoiding injecting into the area and rotating sites can improve glycaemic control and reduce the lipohypertrophy. One study showed that haemoglobin A_{1c} fell from 7.9% to 7.0% in three months after such measures, with a significant reduction in insulin requirement (I Franzen and J Ludvigsson, International Diabetes Federation congress, Helsinki, 1997). Indeed, patients should be advised to reduce their insulin doses once they begin to rotate injection sites, as they may develop hypoglycaemia resulting from improved insulin absorption. Changing treatment to the more rapid acting insulin lispro has been reported as causing less lipohypertrophy, as the insulin is more rapidly absorbed and hence the adipocytes spend less time exposed to the lipogenic actions of insulin.[8]

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